

A possible path for mercury in biological systems: the oxidation of metallic mercury by molecular oxygen in aqueous solutions

Maria Elizabeth Afonso de Magalhães, Matthieu Tubino*

*Instituto de Química, Universidade Estadual de Campinas — UNICAMP, CP6154, Cidade Universitária 'Zeferino Vaz',
Barão Geraldo, Campinas SP, CEP13083-970, Brazil*

Received 19 August 1994; accepted 5 January 1995

Abstract

Metallic mercury has been assumed by several authors as not very reactive and, as a consequence, with little or no toxicity. The toxicity of this element is usually ascribed to alkyl-mercury ions considered to be formed by some microorganisms. In this work, we describe experiments that clearly show that metallic mercury can be easily oxidized by molecular oxygen in aqueous solution in the presence of species such as chloride, which complex Hg(II). The experiments were carried out using metallic mercury in NaCl aqueous solution under 'open air' (temperature and agitation rate maintained constant) and under more controlled conditions (CO₂ rate bubbling, i.e. pH = 4.2; air rate bubbling, i.e., O₂ constant concentration, temperature, agitation rate). The reactions were monitored spectrophotometrically at 230 nm (HgCl₄²⁻). Significant values of the concentration of Hg(II) in the form of HgCl₄²⁻ were soon attained in those solutions. For example, in 'open air' conditions, at 25°C and [NaCl] = 30 g/l (0.51 mol/dm³), the maximal concentration of 13 ppm (6.44 × 10⁻⁵ mol/dm³) of Hg(II) in the form of HgCl₄²⁻ was reached in 120 min; for [NaCl] = 5 g/l at 25°C, (0.085 mol/dm³) the maximal concentration of 0.3 ppm (1.53 × 10⁻⁶ mol/dm³) of Hg(II) in the form of HgCl₄²⁻ was reached in 10 min. The rate constants, *k*_{obs}, of the oxidation of the metallic mercury under the studied conditions are pseudo zero-order at 25°C, and under more controlled conditions have ranged from 1.0 × 10⁻⁷ mol/min ([NaCl] = 5 g/l ≡ 0.085 mol/dm³) to 20.0 × 10⁻⁷ mol/min ([NaCl] = 300 g/l ≡ 5.12 mol/dm³). The rate constant increases with temperature, up to 25°C, from where *k*_{obs} remains constant up to 40°C. From the analysis of the experimental results it was possible to propose a mechanism of oxidation of metallic mercury by O₂ in aqueous solution containing NaCl. This oxidation is proposed as a possible route for the introduction of mercury into biological systems.

Keywords: Mercury; Dissolution; Kinetics; Mechanism; Toxicology; Oxidation

* Corresponding author.

1. Introduction

The environment and man have been sharing the responsibility for mercury production [1]. As industrialization has proceeded, mercury has been intensively mined and released [2]. In 1966 the world production of mercury was estimated to be 9200 tons [3]. Its major uses were in plastics, chlorine and caustic soda production, as well as agricultural usage as fungicides and bactericides [3]. At that time, 4000–5000 tons of mercury per year were discharged into the environment [3,4]. The World Health Organization (WHO) estimated the actual total global release of mercury to the environment due to human activities to be of the order of 2000–3000 tons/year [5]. Historically it has been estimated that the release of mercury to the atmosphere, between 1580 and 1990, because of the precious metal production in the Americas totalled ~ 257 400 tons [6]. These figures are compared by Nriagu [6] with the present global emission of 910–6200 tons/year from a wide array of industrial sources.

It was not until the late 1950s that low level mercury toxicity to humans was noted. Families living in fishing villages along Minamata Bay, Japan, were stricken by a mysterious disease, with a set of symptoms including constriction of the visual fields, progressive weakening of the muscles, numbness, paralysis and, in several cases, coma and death [5,7]. Subsequent investigations identified the so called ‘Minamata disease’ to result from severe mercury poisoning, caused by the large amount of methyl mercury discharged into the bay from a nearby plastic factory [7,8]. The fish from the bay, the main protein source in the daily diet for local people, contained 5–20 ppm of mercury. According to the United States Food and Drug Administration the allowed mercury concentration in fish is 0.5 ppm [8]. The Minamata case is still under investigation and decontamination processes are under way [9].

Other incidents have kept mercury in the news. There have been outbreaks of mercurial poisoning in Iraq, Pakistan, Guatemala and Japan, with hundreds of deaths [7,8], which resulted from eating mercury-contaminated fish or the consump-

tion of foods prepared from seed grain treated with mercurial fungicides [8,10].

Mercury is released into the environment not only in the highly toxic organic forms [11] but also in the elemental form or as inorganic compounds. Its physical/chemical properties and industrial/commercial uses have diversified mercury exposure. Actually, mercury uses have been reduced all over the world [10], so direct acute mercury exposure is now limited to certain occupations, such as the production of electrolytic chlorine, electrical apparatus, catalysts, thermometers, paints and dental amalgam [12]. Mercury amalgamation was the basis for gold extraction [8] in the 18th century in Brazil [13] and the consequences of its release to the surroundings, such as the birth of anencephalic children, are reported even today.

This metal is still used by many ‘garimpeiros’ (surface miners) in the Amazon region [14,15]. In this case, the mercury is discharged into the local environment. It is assumed by some authors [14,15] that 55% of the mercury is released to the atmosphere as metallic vapor (Hg^0), during the ‘frying’ of the Au-Hg alloy, that is frequently carried out in this area without the use of retorts. The rest is probably lost directly to the rivers during the amalgamation process, in the metallic liquid form [14,15]. Thus, it becomes both an occupational hazard and a serious environmental problem [16]. It has been estimated that 70–100 tons of mercury are discharged into the atmosphere and that 30–60 tons are released into the soil and the rivers annually [14,15,17]. More recent estimates suppose that ~ 200 tons/year of mercury are released into the Amazon ecosystem by the activities of 350 thousand ‘garimpeiros’ [18]. This released metallic mercury can contaminate not only the miners themselves but also the inhabitants on the borders of the Amazonian rivers and lakes, and the Indians for whom fish is an important part of their diet. In a recent publication, Veiga et al. [19] proposed that mercury pollution in the Amazon region is mainly a consequence of deforestation by fires. They estimated the mercury emission caused by this process as 710 tons in 1991. Independent of which source of

emission is the more important, the 'garimpeiros' or the fires, this observation clearly shows that the release of mercury in the Amazon region is higher than originally supposed. If the estimated mercury released by the 'garimpeiros' is added to that released from burning of the forests, a total of ~ 900 tons/year is estimated, i.e. approximately one third of the estimated world release [5] of 2000–3000 tons/year to the environment.

The Brazilian press has publicized, in a general way, the drastic effects on the health of the 'garimpeiros' that use(d) metallic mercury to extract gold. These incidences of metallic mercury poisoning clearly show that there exist ways by which this element is 'chemically' introduced to the human body. We plan to show that from our point of view, this introduction is mainly accomplished after oxidation of the metallic mercury to Hg(II).

It has been pointed out by Pryde [8] that methyl mercury, (CH_3Hg^+) , and dimethyl mercury, $(\text{CH}_3)_2\text{Hg}$, are the toxic forms of mercury responsible for permanent injuries. In particular, the human body can not efficiently metabolize monomethyl mercury and it remains in the body for a relatively long time (biological half-life of 70 days) [8].

It is interesting to note that, in fish, mercury is found almost entirely in the form of methyl mercury compounds despite the fact that most mercury released into rivers, lakes and oceans, is in the elemental form or as inorganic salts [20].

Mercury has a strong attraction for sulfur [8,21] found in sulfur-hydrogen linkages in proteins (especially enzymes), and it is bound in cell membranes, altering the cell's proper functions [8,21], mainly the sensory and motor functions of the central nervous system [21,22], causing biological damage [8]. The affinity by sulfur- and nitrogen-containing molecules is not only a characteristic of the methyl-mercury cation, but of mercury itself [23]. It is easy to suppose that the affinity of mercury for proteins is evidence that, after oxidation of this element in organic systems, it can be rapidly linked to proteins present and, in this way, be transported into the biological system.

The toxicity of mercurials for man varies greatly with the form in which it is taken into the body,

as well as with the route by which it enters. Goldwater and Clarkson [20] have pointed out that there is a failure to understand the conversion of the relatively 'innocuous' metallic mercury into 'toxic' methyl mercury. The methylation process has been studied for many years because it is a mechanism that plays an important role in the mobilization and transport of the metal [24,25] in the environment.

According to Clarkson [26] and Manahan [27], when inhaled, elemental metallic mercury vapor, being relatively diffusible [12], is readily absorbed across the alveolar membranes. Hayes and Rothstein [28] believe that the elemental mercury attached to lung tissue is oxidized to inorganic mercury ions. Both Koos and Longo [12] and Clarkson [26] state that the mercury that crosses the lung membranes in the elemental form has high diffusibility and lipid solubility, leading to rapid penetration across cell membranes. According to Koos and Longo [12], both mercurous ions and metallic mercury are rapidly converted to mercuric ions by intracellular enzymes. But in the work of Clarkson [26], Hursh et al. [29] and Langworth [30] the oxidation of the elemental mercury to the divalent ion appears to take place in the red blood cells and in some tissues. The inorganic mercurials bind to serum albumin, plasma and to hemoglobin [12,20].

Brain levels were shown by Goldwater and Clarkson [20] to be higher after exposure to mercury vapor, since there will always be small amounts of dissolved vapor in the blood stream during the exposure period. They suppose that the high lipid solubility and lack of charge permit the dissolved vapor to readily cross the blood brain barrier. The vapor is then oxidized in the brain since mercury, once deposited in this organ, is very slowly released [20].

Although Koos and Longo [12] pointed out that metallic mercury is not appreciably absorbed by the human gastrointestinal tract and oral ingestion of metallic mercury has been then considered harmless, diarrhea and stomatitis were observed [12]. Mercury also can be absorbed through the skin, but this process is too slow to be of much importance except in unusual circumstances [12]. The mechanism of skin absorption is

not well established. Studies found in the literature on rates of skin absorption of elemental mercury are rare and imprecise [26,31]. For example, under conditions of occupational exposure, it is difficult to determine the proportion of mercury absorbed through the skin and by inhalation [26].

Unlike metallic mercury, mercuric ions (Hg^{2+}) are more readily absorbed through the gastrointestinal tract. Approximately 5–15% of the total amount ingested is absorbed via this route. Mercurous ions (Hg_2^{2+}) are less water soluble than Hg^{2+} compounds; therefore, they are not as well absorbed orally. It is considered that inhaled aerosols of mercuric salts are absorbed by the lungs, although not as well as metallic mercury vapor. Mercuric ions can also be absorbed by the skin, but less so than by other routes [12].

The tissues containing the greatest concentration of mercury after acute exposure are kidney, liver, spleen, muscle and blood [12].

To react chemically with proteins and other molecules in living organisms, elemental mercury must undergo oxidation to the mercurous (Hg_2^{2+}) or mercuric (Hg^{2+}) ion. Since the general pattern of distribution in body tissues is similar following exposure to vapor of elemental mercury and after injection of the divalent cation, oxidation of the vapor probably takes place soon after absorption from the lungs [26]. The interaction of mercury with blood, for example, seems to be dependent on an oxidation of the metal [30]. Little mercury vapor is taken up by blood in the absence of oxygen [28] and the uptake rate of mercury is dramatically influenced by the chemical composition of the solution [26].

The solubility of metallic mercury in water has been measured by a variety of experimental techniques [32,33]. The recommended value of the solubility of mercury in air-free water is $(3.03 \pm 0.12) \times 10^{-7}$ mol/kg, ~ 60 ppb at 298.15 K [33]. Other values [34] of the solubility of mercury in pure water are close to the recommended value. Sanemasa [35] and Sanemasa et al. [36] did not consider the problem of air oxidation of mercury in their experiments and, even though the solubility value they obtained at 298 K was very close to the recommended value, in temperatures below

and above 298 K their data were different from the experimental values reported by other authors [33]. Crude thermodynamic calculations indicate that oxygen partial pressures as low as 10^{-3} bars could oxidize enough mercury to mercury(II) in aqueous solution to affect the solubility results [33]. This situation seems to be related to a kinetic aspect [33]. In polarography studies, Kolthoff [37] observed that when halides are present in sufficient concentration, relatively small amounts of oxygen in the solution oxidize mercury. Stock et al. [38] also observed the oxidation of metallic mercury in aqueous salt solutions. Several papers report the solubility of mercury in aqueous electrolyte solution [33]. All of the values are classed as tentatives [33], and were carried out at different salt concentrations and range of temperature [35,36,39,40]. Sanemasa [35] and Sanemasa et al. [36] reports were acquired in 'open air' conditions, and the value for the mercury solubility is < 60 ppb (value in absence of oxygen [33]) at 298 K for NaCl sea water concentration. Glew and Hames [39] reported a lower value of 3.3 ppb in NaCl saturated solution for the same temperature. For sea water concentration the values are consistent with the salting out properties of the electrolytes [33].

In elemental mercury toxicity, the crucial chemical step is the oxidation of zerovalent mercury to the divalent mercuric ion [41]. Once oxidized, methylation of the mercuric ion is readily achieved [42]. The methyl mercury cation, once formed, complexes easily with amino acids [43] and other body and environmental fluids [8,21,22].

Less attention has been paid to the importance of the mercuric ion that results from the oxidation of the metallic mercury and can then complex with nucleosides [44], with thiol groups [45], with glycine [46] and with other amino acids under physiological conditions [47]. When chloride ions are present to simulate physiological fluids [46,47] there is a competition in the complexation because chloride is bound to mercury(II) ions with high stability [46,47]. There is a need for a better understanding of the $\text{Hg(II)}/\text{chloride}$ system [46,47].

From the above review of the literature one can envisage the large amount of information

that already exists concerning the toxicology of mercury and related topics. However, the pathways of introduction of mercury into biological systems are not very well understood as yet. The present work was carried out with the aim of contributing to this understanding.

2. Experimental

Spectrophotometric methods for mercury(II) determination in aqueous chloride solutions are very simple and sensitive. The spectra of the HgX_4^{2-} complexes (where X is Cl^- , Br^- , I^-) have been known for many years [48]. The determination of mercury based on UV absorption of HgCl_4^{2-} or HgBr_4^{2-} has been used for environmental samples [49].

Kinetic experiments using the apparatus shown in Fig. 1 were performed with and without CO_2 and air bubbling to follow mercury dissolution by HgCl_4^{2-} formation. This UV-spectrophotometric determination was used for monitoring the reaction in a HP8452 UV/VIS Diode-Array Spectrophotometer. The temperature was controlled by circulating thermostated water around the reaction vessel; the salt solution was kept under controlled agitation, by the use of a PTFE-coated

magnetic bar, resulting in a splitting of the metallic mercury into little droplets of approximately the same size. It has been shown by ultraviolet spectroscopy that tetrachloromercurate ions, HgCl_4^{2-} , are the predominant species in solutions of mercuric chloride in aqueous alkali metal chloride, when the latter is in excess [48].

2.1. Materials and methods

To prepare the salt solutions, analytical grade NaCl was used. The metallic mercury was of analytical grade, but to prevent interferences, it was washed, under an air flow, with a 10% HNO_3 solution to promote oxidation and further dissolution of any metallic interferences. Then, to guarantee the purity of the mercury, the metal was dried and distilled under low pressure. In the reaction vessel, 4.0 g of this treated metallic mercury were put into contact with 40.0 ml of the aqueous NaCl solution. This aqueous solution was monitored by continuous circulation through an optical quartz circulation cell, 1 cm path length, that was placed into a thermostated holder in the spectrophotometer. The wavelength was 230 nm, suitable for HgCl_4^{2-} spectrophotometric determination. The data were acquired every 1 or 2 min during at least 2 h. The first kinetic data were obtained

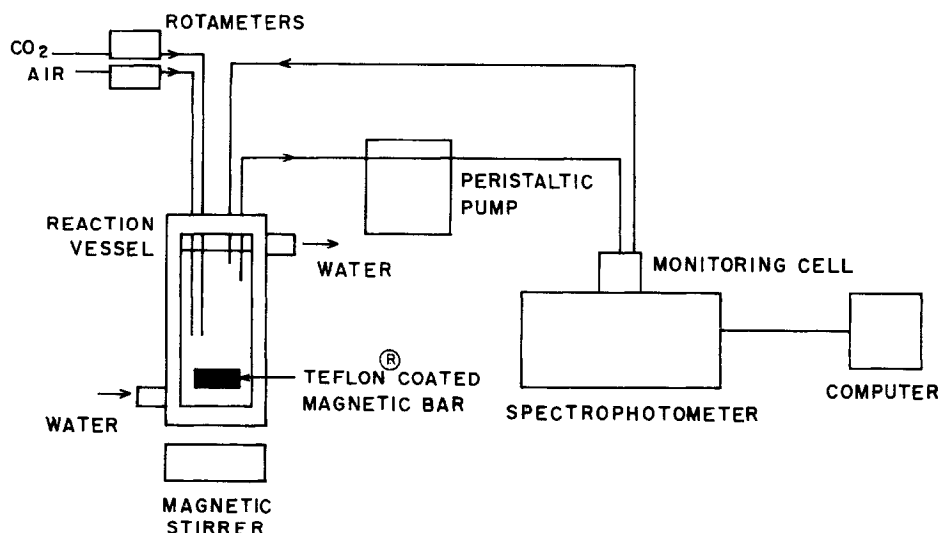


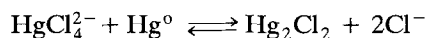
Fig. 1. Layout of the experimental apparatus used for the kinetics runs.

without any control of the pH and of the oxygenation of the solution, i.e. without bubbling CO_2 or air. When CO_2 and air were introduced, a rotameter was used to better control the gas volumes. Analytical grade CO_2 , from a cylinder, was bubbled at a rate of 3 l/h, leading to a constant pH value of 4.2. The air was bubbled in from a membrane bomb, attached to a voltage controller, at the rate of 4.5 l/h. Calibration curves were made using HgCl_2 dissolved in two different concentrations of NaCl solutions (30 g/l or 0.51 mol/dm³ and 150 g/l or 2.55 mol/dm³). No difference was observed for the molar absorptivity of the HgCl_4^{2-} at these two different salt concentrations. Beer's law was obeyed up to 6×10^{-5} mol/dm³ and the molar absorptivity was determined as $(2.55 \pm 0.01) \times 10^4$ l/mol/cm.

The observed constant value was taken from the first 11 min of the reaction, discarding the two initial minutes because of the imprecision of these data. Therefore, a 9-min interval was used for calculation of the constant, using a pseudo zero-order in the kinetic law. This procedure improves the reproducibility of the kinetic constant.

3. Results and discussion

The kinetic runs performed without bubbling CO_2 and air gave characteristic kinetic curves as shown in Fig. 2. Mercury(II) concentration achieves a maximum with a later decrease. This diminution in HgCl_4^{2-} concentration can be ascribed to the reduction of the Hg(II) to Hg(I) according to the well known reaction:



This supposition is supported by the observation that the metallic mercury droplets lost their brightness, becoming white and the characteristic coalescence of the metal did not occur after agitation was stopped.

The maximum absorbance observed in Fig. 2 corresponds to ~13 ppm (6.44×10^{-5} mol/dm³) of mercury. It was verified in our experiments that the concentration of mercury depends on the chloride concentration and the temperature of the solution.

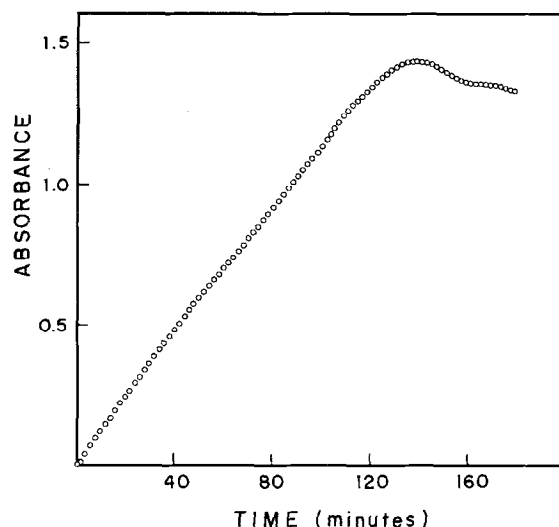


Fig. 2. Kinetic spectrophotometric ($\lambda = 230$ nm) monitoring of the formation of HgCl_4^{2-} from Hg^0 in an aqueous solution of NaCl ($[\text{NaCl}] \approx 30$ g/l ≈ 0.51 mol/dm³), without bubbling CO_2 and air, at $25.0 \pm 0.1^\circ\text{C}$.

Fig. 3 demonstrates the pseudo zero-order rate constant values, showing that the dissolution of metallic mercury is almost linearly proportional to the NaCl concentration, increasing with the concentration of the salt up to ~200 g/l (3.42

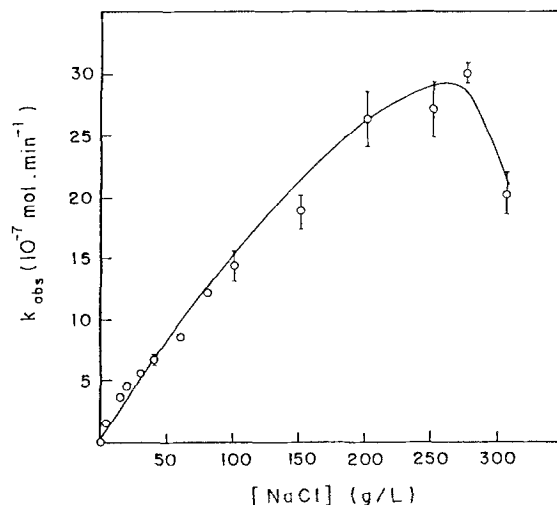


Fig. 3. Pseudo zero-order rate constant for the formation of HgCl_4^{2-} from Hg^0 in aqueous solutions of NaCl as a function of the NaCl concentration.

mol/dm³) when an unexpected rapid decrease is observed. This decrease, as will be shown later, can be ascribed to a significant decrease in the oxygen concentration [50] below 2 ppm (6.25×10^{-5} mol/dm³). The increase of k_{obs} as a function of increasing salt concentration clearly shows the importance of chloride in the mechanism of metallic mercury dissolution.

Experiments where pH was also monitored using a glass electrode clearly showed H⁺ consumption with time, changing from an initial value of pH = 5.3 to a final, and constant, value of pH = 8.6. From these experiments, it can be concluded that the reaction is highly dependent on the H⁺ concentration. In fact, it was observed that the addition of a few drops of a 0.1-mol/dm³ HCl solution significantly increases the rate constant.

To maintain the oxygen concentration in the solution constant, air was bubbled into the solution. A membrane pump was used and to avoid fluctuations, due to heating of the pump, an external voltage controller was used. The flow rate was measured by a rotameter (4.5 l/h). To maintain the pH of the solution constant (pH = 4.2), analytical grade CO₂ from a cylinder was bubbled simultaneously with the air. Its flow rate was controlled by a rotameter (3.0 l/h). The polyethylene (i.d. \approx 1 mm) tubes conducting the gases were introduced a little below the half height of the solution (Fig. 1).

Instead of air, O₂ could be used. However, air bubbling gave adequate results. From the observed results the importance of both the oxygen and pH in the reaction performance is very clear.

The relation between the observed rate constant for mercury dissolution and the salt concentration with CO₂ and air bubbling is shown in Fig. 4.

It is interesting to compare Fig. 3 with Fig. 4. When CO₂ and air are not bubbled into the solution, higher values are achieved for the rate constant, $\sim 30 \times 10^{-7}$ mol/min. When bubbling is used, the observed rate maxima are $\sim 20 \times 10^{-7}$ mol/min. This difference can be understood on the basis of a higher initial total O₂ concentration in the solution than is the case when CO₂ is not introduced. On the other hand, when the

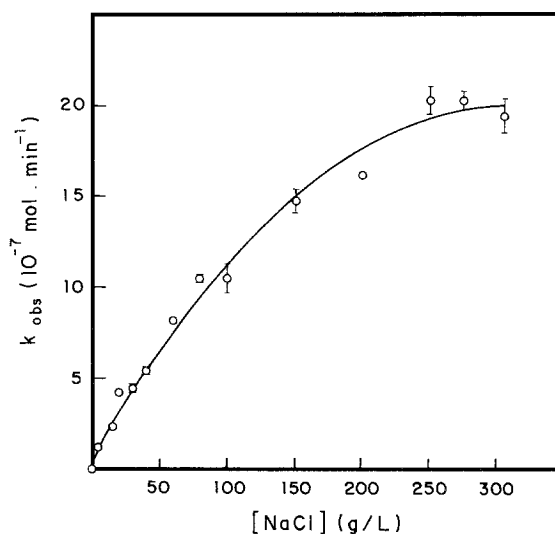


Fig. 4. Pseudo zero-order rate constant for the formation of HgCl_4^{2-} from Hg^0 in aqueous solutions of NaCl, with CO₂ and air bubbling, correlated to the NaCl concentration.

NaCl concentration is 300 g/l (5.13 mol/dm^3), a decrease is observed in Fig. 3, but not in Fig. 4, where air is continuously introduced into the solution. This can be easily ascribed to exhaustion of the oxygen in the former case (Fig. 3). According to MacArthur [50], who studied the solubility of molecular oxygen in aqueous solutions of NaCl from 7.3 g/l (0.12 mol/dm^3) up to $\sim 240 \text{ g/l}$ (4.10 mol/dm^3), the O₂ concentration decreases from $\sim 8 \text{ ppm}$ ($0.25 \times 10^{-3} \text{ mol/dm}^3$) to 2.3 ppm ($7.2 \times 10^{-5} \text{ mol/dm}^3$). On comparing these data with those of Figs. 3 and 4, it can be supposed that the decrease in the rate constant in Fig. 3, and the non-linearity of the curve in Fig. 4, are consequences of the decrease in the oxygen concentration in the solution as a function of the increase in the salt concentration. This supposition is also in accordance with the observed results of the dependence of the rate constant as a function of the temperature, as shown in Fig. 5. Between 10°C and 25°C, within experimental error, the reaction follows the Arrhenius ($E_a = 21.8 \text{ kJ/mol}$) and Eyring ($\Delta H^\ddagger = 19.4 \text{ kJ/mol}$; $\Delta S^\ddagger = .94 \text{ J/mol K}$) relations. Above this temperature (up to 40°C), k_{obs} tends to a constant value. This can be related again to a decrease in

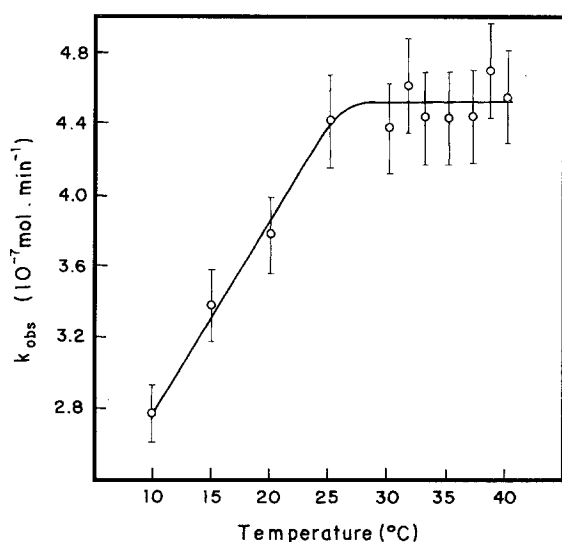


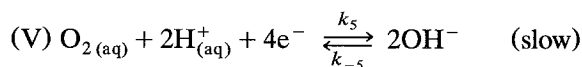
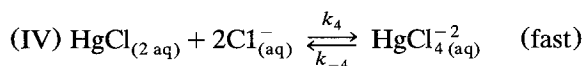
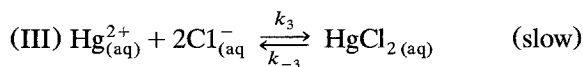
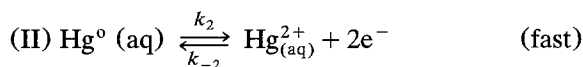
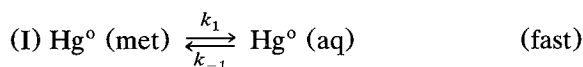
Fig. 5. Observed rate constant as a function of the temperature, in a 30-g/l (0.51 mol/dm³) NaCl solution.

the oxygen concentration in the solution as a function of temperature [51].

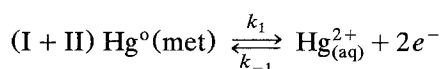
4. Conclusions

Despite the fact that the oxidation of metallic mercury in aqueous solution of ions such as chloride was reported ~ 50 years ago by Kolthoff [37] and ~ 60 years ago by Stock et al. [38], very little attention has been paid to this reaction in the literature concerned with toxicology, ambient environmental problems, occupational exposure, etc., related to this element. From the results obtained in this work it is possible to correlate the phenomenon of oxidation and dissolution of this metal to several of these problems.

Initially, the mechanism¹ of dissolution and oxidation of metallic mercury can be envisaged as:



On the other hand, it could be considered that the oxidation of metallic mercury occurs on the surface of the droplets, with subsequent dissolution of the oxidized element and not in the solution as shown by paths I and II of the previous mechanism. Actually it is not possible to differentiate whether the oxidation occurs on the metallic surface or after the dissolution of the metallic mercury. Both processes are possible and, probably, both are in effect. Experimentally, it was observed that the rate depends on agitation² and on the size of the droplets and this reinforces both possibilities. The agitation not only facilitates dissolution of the metallic mercury but also renovation of the metallic surface, increasing the contact surface by decreasing the droplet size. If the mercury oxidation is considered as occurring on the metallic surface, Eqs. I and II become:



Independent of whether it is reactions I and II or (I + II) which are effective, the equilibrium between $\text{Hg}_{(\text{met})}^0$ and $\text{Hg}_{(\text{aq})}^{2+}$ must be very rapid, relative to the others, and $[\text{Hg}_{(\text{aq})}^{2+}]$ should be in a steady-state concentration.

Based on this reaction scheme, and considering

¹Considering that the monitored specie is HgCl_4^{2-} , the criteria used to decide whether an equilibrium (I–V) is ‘fast’ or ‘slow’ relative from one to another were as follows: (1) the total process (I–V) is dependent on the concentrations of Cl^- , O_2 and H^+ . Therefore, the equilibria involving these species are relatively slow, i.e. V and III and/or IV. As HgCl_2 is not spectrophotometrically observed during the monitoring of the reaction it can be considered that the equilibrium III is the slow one and therefore IV is fast; (2) the equilibria I and II must be rapid because if they were slow relative to III and V, the total process couldn’t be dependent on the Cl^- , O_2 and H^+ concentrations.

²The increasing or decreasing of the rate by variations in the agitation of the metallic mercury is a consequence of the variation of $[\text{Hg}_{(\text{aq})}^{2+}]$ and therefore of the rate of the path III. As the $[\text{Hg}_{(\text{aq})}^{2+}]$ is not known, and it was ‘included’ in k_{obs} values, apparent variations in this data can be observed.

$k_4 \gg k_5$, the following rate law can be deduced³, considering that the monitored specie is $\text{HgCl}_{4(\text{aq})}^{2-}$:

$$\begin{aligned} d[\text{HgCl}_{2(\text{aq})}]/dt &= d[\text{HgCl}_{4(\text{aq})}^{2-}]/dt \\ &= \underbrace{k_3 [\text{Hg}_{(\text{aq})}^{2+}][\text{Cl}^-]^2}_A - \underbrace{k_{-3} [\text{HgCl}_2]}_B \end{aligned} \quad (1)$$

Well before attaining equilibrium, ($\nu_3 \gg \nu_{-3}$), $A \gg B$ and the equation can be simplified as:

$$d[\text{HgCl}_{4(\text{aq})}^{2-}]/dt = k_3 [\text{Hg}_{(\text{aq})}^{2+}][\text{Cl}^-]^2 \quad (1a)$$

As $[\text{Hg}_{(\text{aq})}^{2+}]$ can be considered constant due to the conditions of the experiments (agitation, mercury mass, size of the mercury droplets, etc.), and the chloride concentration is fixed then, $k_3 [\text{Hg}_{(\text{aq})}^{2+}][\text{Cl}^-]^2 = \text{constant} = k_{\text{obs}}$ (zero-order reaction). Consequently:

$$d[\text{HgCl}_{4(\text{aq})}^{2-}]/dt = k_{\text{obs}} \quad (2)$$

Indeed, k_3 and, therefore, k_{obs} , also includes $[\text{O}_{2(\text{aq})}]$ and $[\text{H}_{(\text{aq})}^+]$. As the hydrogen ion concentration is maintained constant by CO_2 bubbling, in our experiments, the observed rate constant is only dependent on the chloride concentration. When $[\text{O}_2]$ and/or $[\text{H}^+]$ change, a corresponding variation in the rate is observed.

The observed metallic mercury dissolution/oxidation clearly shows that this reaction can be considered a means of entry of this element into the environmental cycle. Certainly, if other inorganic or organic complexing species are present, instead of chloride, a similar reaction of oxidation is expected. Experiments carried out in our laboratory with bromide and with iodide show similar processes where higher k_{obs} values have been observed, as expected from the values of the relevant equilibrium constant of the $[\text{Hg}(\text{X})_4]^{2-}$ complexes ($\text{X} = \text{Cl}^-$, Br^- and I^-), being $\sim 8 \times 10^{-16}$ for Cl^- , 1×10^{-21} for Br^- and 1.5×10^{-30} for I^- [52] at 25°C and 0.5 ionic strength.

³ It must be noted that the rate of formation of HgCl_4^{2-} is effectively equal to the rate of formation of HgCl_2 as process III is slower than IV and therefore the amount of the HgCl_4^{2-} that can be formed depends on the concentration of HgCl_2 that reacts.

Based on these aspects, it should be possible to observe several situations where metallic mercury can be oxidized, in the human (or animal) body, in chlor-alkali factories, in sea water, in river water and in the environment in general.

In the human body, for example, various situations can be supposed. In the stomach there is a considerable chloride concentration as a result of the hydrochloric acid present as well as from food. The temperature (37°C) and the pH are also favorable and oxygen is present. Therefore, oxidation of swallowed metallic mercury is quite favorable and the formation of HgCl_4^{2-} is expected. As is widely known, this compound forms precipitates of basic salts and/or oxides of mercury(II) in alkaline media and, consequently this transformation is expected to occur in the intestinal tract. Therefore, absorption of mercury under these conditions is probably not favored. This supposition is in accordance with Koos and Longo [12] who pointed out that mercury is not appreciably absorbed by the human gastrointestinal tract.

The intake of metallic mercury by the respiratory system also favors the oxidation of this element, as in arterial blood (pH ≈ 7 and 37°C) the oxygen concentration is relatively high and there are also present organic and inorganic species, including Cl^- , that can complex Hg^{2+} . Inhalation of metallic mercury also favors oxidation due to the high superficial area of contact of the vapor. Absorption of mercury through alveolar membranes has been reported [12,26,27] and the oxidation of mercury in blood cells and in some tissues appears to take place [26,29,30].

Contact of metallic mercury with the skin has usually been considered as non-problematic. However, as body perspiration contains chloride and its evaporation on the skin surface increases the salt concentration, oxidation of mercury is expected. There is no evidence suggesting whether or not there is absorption by the skin following this oxidation but, at least, mercury can be released to the environment in the ionic form. Koos and Longo [12] considered that mercury is only slowly absorbed by the skin under normal circumstances.

Following the introduction of the HgCl_4^{2-} into the body, its transformation into organo-mercurial

compounds under physiological conditions can be envisaged. Mercury(II) salts in the presence of, for instance, amino-acids slowly exchange chloride for the organic ligand. Maeda et al. [47] have observed this reaction. Reaction with amino-acids in blood obviously suggests ways in which mercury can spread throughout the body.

The oxidation of metallic mercury in sea water and in concentrated aqueous solutions of NaCl in factories producing chlorine and hydroxide is obvious. It is also easy to envisage oxidation of atmospheric metallic mercury if present in sea water spray which is highly concentrated in chloride and other anions. However, slow conversion of Hg^0 to Hg(II) can be expected under atmospheric conditions if a complexing agent, as for instance Cl^- , is absent. This is in agreement with the estimated residence life time of Hg^0 in atmosphere that is measured in months or years so that, once released into the atmosphere, mercury vapor is globally distributed [53]. Schroeder [54] had already calculated the distribution of major inorganic mercury species in sea water, and divalent mercury, Hg(II) , should exist in sea water, predominantly as tetra-chloro complex. The oxidation velocity would depend of the natural conditions, but as shown in the present work it could be much faster than already supposed.

In the case of chlor-alkali factories it can be supposed that the spray from the NaCl solutions could contain HgCl_4^{2-} that can be absorbed by workers lungs and introduced directly into the blood.

In the Amazon region, where waters are relatively rich in organic matter, one can imagine the oxidation of elemental mercury to form organo-mercury complexes. This situation should be more favorable in estuarine water.

Once mercury is oxidized and forms organo-mercury complexes in blood, their attachment to several tissues of the body, causing damage, is an obvious consequence. In addition, the organo-mercury complexes (as well as inorganic complexes) that can be formed in rivers, lakes and sea water are probably easily absorbed by the local biosphere, initiating the bio-concentration of mercury through successive trophic levels and the human food chain.

5. Final comments

The usual statement that metallic mercury is innocuous or little toxic needs to be reviewed. As suggested by this work the metal can enter the biological cycle if the appropriated conditions are present, i.e. molecular oxygen, chloride or other complexing specie and $\text{pH} < 8$. Even being immobilized, for example, in the bottom of bays, the metallic mercury could slowly react to form HgCl_4^{2-} , and enter the food chains. The process needs to be looked at from a global point of view.

References

- [1] C.E. Knapp, Mercury in the environment, *Environ. Sci. Technol.*, 4 (1970) 890–892.
- [2] D. Lloyd, Mercury: anatomy of a pollution problem, *Chem. Eng. News*, 49 (1971) 22–34.
- [3] D.H. Klein and E.D. Goldberg, Mercury in the marine environment, *Environ. Sci. Technol.*, 4 (1970) 765–768.
- [4] W.F. Fitzgerald and T.W. Clarkson, Mercury and monomethylmercury: present and future concerns, *Environ. Health Perspect.*, 96 (1991) 159–166.
- [5] WHO, Environmental Criteria 1: Mercury, 101: Methylmercury and 118: Inorganic Mercury, World Health Organization, Geneva, 1991.
- [6] J.O. Nriagu, A precious legacy, *Chem. Br.*, 30 (1994) 650–651.
- [7] J. Bockris, *Environmental Chemistry*, Plenum Press, New York, 1977, pp. 452–460.
- [8] L.T. Pryde, *Environmental Chemistry, An Introduction*, Cummings Publishing Company, Menlo Park, California, 1973, pp. 200–206.
- [9] A. Kudo and S. Miyahara, A case history; Minamata mercury pollution in Japan — from loss of human lives to decontamination, *Water Sci. Technol.*, 23 (1991) 283–290.
- [10] H.S. Stoker and S.L. Seager, *Environmental Chemistry: Air and Water Pollution*, Scott, Foresman and Company, Glenview, 1972, pp. 119–130.
- [11] L. Hodges, *Environmental Pollution*, Rinehart and Winston, New York, 1973, pp. 422–424.
- [12] B.J. Koos and L.D. Longo, Mercury toxicity in the pregnant woman, fetus and newborn infant, *Am. J. Obstet. Gynecol.*, 126 (1976) 390–409.
- [13] P. Bertran, Desastres ambientais na capitania de Goias, *Ciência Hoje*, 12 (1991) 42–48.
- [14] L.D. Lacerda and W.C. Pfeiffer, Mercury from gold mining in the Amazon environment — an overview, *Quim. Nova*, 15 (1992) 155–160.
- [15] W.C. Pfeiffer et al., A ameaça do mercúrio nos garimpos, *Ciência Hoje*, 11 (1990) 10–12.
- [16] L. Byrne, Brazil's mercury poisoning disaster, *Br. Med. J.*, 304 (1992) 1397.

- [17] P. Grandjean, B. Cardoso, G. Guimarães, Mercury poisoning, *The Lancet*, 342 (1993) 991.
- [18] A. Petry et al., *Em busca do ouro limpo*, *Veja*, 27 (1994) 75.
- [19] M.M. Veiga, J.A. Meech and N. Onate, Mercury pollution from deforestation, *Nature*, 368 (1994) 816–817.
- [20] L.J. Goldwater and T.W. Clarkson, Mercury, in D.H.K. Lee (Ed), *Environmental Sciences*, Academic Press, New York, 1972, pp. 17–55.
- [21] A.J. Carty and S.F. Malone, The chemistry of mercury in biological systems, in: J.O. Nriagu (Ed), Vol. 3, *Topics in Environmental Health*, Elsevier, Amsterdam, 1979, pp. 433–479.
- [22] R.P. Junghans, A review of the toxicity of methylmercury compounds with application to occupational exposures associated with laboratory uses, *Environ. Res.*, 31 (1983) 1–31.
- [23] F.A. Cotton and G. Wilkinson, *Advanced Inorganic Chemistry*, Wiley-Interscience, New York, 1972.
- [24] S. Krishnamurthy, Biomethylation and environmental transport of metals, *J. Chem. Educ.*, 69 (1992) 347–350.
- [25] S. Jensen and A. Jernelev, Biological methylation of mercury in aquatic organisms, *Nature*, 223 (1969) 753–754.
- [26] T.W. Clarkson, The pharmacology of mercury compounds, *Annu. Rev. Pharmacol.*, 12 (1972) 375–406.
- [27] S.E. Manahan, *Toxicological Chemistry*, Lewis, Chelsea, Michigan, 1991, p. 104.
- [28] A.D. Hayes and A. Rothstein, The metabolism of inhaled mercury vapor in the rat studied by isotope techniques, *J. Pharmacol. Exp. Therap.*, 138 (1962) 1–10.
- [29] J.B. Hursh, S.P. Sidrak and T.W. Clarkson, In vitro oxidation of mercury by the blood, *Pharmacol. Toxicol.*, 63 (1988) 266–273.
- [30] S. Langworth, *Early Effects of Occupational and Environmental Exposure to Inorganic Mercury*, PhD Thesis, Huddinge University Hospital, Institute of Occupational Medicine, Karolinska Institute, Stockholm, Sweden, 1992.
- [31] J.B. Hursh et al., Percutaneous absorption of mercury vapor by man, *Arch. Environ. Health*, 44 (1989) 120–127.
- [32] V.I. Sorokin, Solubility of mercury in water at temperature from 300 to 500°C and pressure from 500 to 1000 atm, *Dokl. Acad. Nauk. SSSR*, 213 (1973) 852–853.
- [33] H.L. Clever, S.A. Johnson and M.E. Derrick, The solubility of mercury and some sparingly soluble mercury salts in water and aqueous electrolyte solutions, *J. Phys. Chem. Ref. Data*, 14 (1985) 631–680.
- [34] E. Onat, Solubility studies of metallic mercury in pure water at various temperatures, *J. Inorg. Nucl. Chem.*, 36 (1974) 2029–2032.
- [35] I. Sanemasa, The solubility of elemental mercury vapor in water, *Bull. Chem. Soc. Jpn.*, 48 (1975) 1795–1798.
- [36] I. Sanemasa, K. Haraguchi and H. Nagai, Effects of salts on the solubility of elemental mercury in water, *Bull. Chem. Soc. Jpn.*, 54 (1981) 1040–1043.
- [37] I.M. Kolthoff, Factors to be considered in quantitative polarography, *Ind. Eng. Chem.*, 14 (1942) 195–200.
- [38] A. Stock et al., Vaporization, solubility and oxidation of metallic mercury, *Z. Anorg. Allgem. Chem.*, 217 (1934) 241–253.
- [39] N.D. Glew and D.A. Hames, Mercury solubility in 6.10 molal sodium chloride, *Can. J. Chem.*, 50 (1972) 3124–3128.
- [40] V.P. Chviiruk and N.V. Koneva, Solubility of metallic mercury in solutions of electrolytes, *Ukr. Khim. Zh.*, 41 (1975) 1162–1164.
- [41] D.C. Winfield and S. Tse, Kinetics and mechanism of the oxidation of mercury by peroxidase, *Can. J. Chem.*, 63 (1985) 2940–2944.
- [42] J.M. Bellama et al., A comparison of the rates of methylation of mercury(II) species in aquatic media by various organotin and organosilicon moieties, *Sci. Total Environ.*, 73 (1988) 39–51.
- [43] S. Alex and R. Savoie, A Raman spectroscopic study of the complexation of the methylmercury(II) cation by amino acids, *Can. J. Chem.*, 65 (1987) 491–496.
- [44] G.L. Eichhorn and P. Clark, The reaction of mercury(II) with nucleosides, *J. Am. Chem. Soc.*, 85 (1963) 4020–4024.
- [45] B.V. Cheesman, A.P. Arnold and D.L. Rabenstein, Nuclear magnetic resonance studies of the solution chemistry of metal complexes, *J. Am. Chem. Soc.*, 110 (1988) 6359–6364.
- [46] S.R. Reid and B. Podányi, A proton NMR study of the glycine-mercury(II) system in aqueous solution, *J. Inorganic Biochem.*, 32 (1988) 183–195.
- [47] M. Maeda, M. Tsunoda and Y. Kinjo, Coordination of mercury to amino acids under physiological conditions, *J. Inorganic Biochem.*, 48 (1992) 227–232.
- [48] G.B. Deacon, Chemistry of halomercurate(II) complexes, *Rev. Pure Appl. Chem.*, 13 (1963) 189–210.
- [49] U.V. Naidu and G.R. Naidu, Spectrophotometric determination of mercury in water, *Acta Chim. Hung.*, 128 (1991) 355–358.
- [50] C.G. MacArthur, Solubility of oxygen in salt solutions and the hydrates of these salts, *J. Phys. Chem.*, 20 (1916) 495–502.
- [51] C.J.J. Fox, On the coefficients of absorption of nitrogen and oxygen in distilled water and sea-water and of atmospheric carbonic acid in sea water, *Trans. Faraday Soc.*, (1909) 69–87, in: M.C. Whipple and G.C. Whipple, Solubility of oxygen in sea water, *J. Am. Chem. Soc.*, 33 (1911) 362–365.
- [52] R.M. Smith and A. Martell, *Critical Stability Constants*, Vol. 4, Plenum Press, New York, NY, USA, 1976.
- [53] T.W. Clarkson, Mercury: major issues in environmental health, *Environ. Health Perspect.*, 100 (1992) 31–38.
- [54] W.H. Schroeder, Developments in the speciation of mercury in natural waters, *Trends Anal. Chem.*, 8 (1989) 339–342.